# Our Thanks to Westwood

*We recognize with appreciation Westwood Pharmaceuticals for pledging support to the Endowment Fund for* The Journal of Investigative Dermatology, *which will be used to support the growth and continued success of the Journal. This support will certainly strengthen and perpetuate the partnership between the pharmaceutical industry and basic and clinical investigators in cutaneous biology.*

*We salute Westwood Pharmaceuticals for their contribution to the Endowment Fund and for their continued support of clinical and investigative dermatology.*

*D.A.N., Denver, CO*



# In This Issue . . .

David A. Norris

We wish to express our thanks to Jean Marx for her contributions to this feature over the past two years. Jean has been named Deputy News Editor of *Science,* and will be unable to continue as author of this feature. "In This Issue . . . " will now be written by members of the Editorial Board.

## New Biochemical Abnormalities in Lamellar Ichthyosis

The normal stratum corneum is the major barrier to external irritants and prevents dehydration. Its normally smooth surface can be disrupted by weather, harsh chemicals, or inflammation. In addition, people with many skin diseases or genetic disorders such as ichthyosis may develop scales or thick plates instead of the smooth, regular stratum corneum. Uncommon diseases such as ichthyosis can provide important clues to the complex structural and biochemical events involved in epidermal differentiation.

The lamellar body (Odland body, membrane coating granule) is an organelle of the upper granular layer. It is rich in lipids and in enzymes which are believed to modify lipid composition, producing the hydrophobic lipids found in the stratum corneum. In this issue, it is shown that different quantities of enzymes present in the lamellar body may provide a means for distinguishing between the two major subsets of lamellar ichthyosis. Mieke Bergers, Sabine Dunnwald, Paul Mier, Renske van Dooren-Grebe, Peter Steijlen, and Rudolph Happle at the University of Nijmegan in The Netherlands, and Heiko Traupe at the University of Munster in West Germany studied scale extracts from nine patients with erythrodermic autosomal recessive lamellar ichthyosis (EARLI), and 12 patients with the nonerythrodermic form (NEARLI). "We were able to differentiate between EARLI and NEARLI by measuring three enzymes and determining their ratios," Dr. Bergers said. The ratios among B-gluconidase, butyrase, and palmitase distinguished EARLI from NEARLI groups and were consistent within families. However, these measurements could not be used to distinguish ichthyotic scale from psoriatic scale. Since these enzymes are necessary for modifying epidermal lipids, the authors propose that differences in their relative proportions may determine the different lipid patterns and abnormal stratum corneum seen in these patients. In contrast to previous work reporting elevated n-alkane levels in EARLI but not NEARLI patients, this approach is not influenced by possible contamination by exogenous material.

More recent work from this laboratory on the differential extractability of these enzymes from ichthyotic scale further supports the concept of enzymatic differences between these two patient groups. Further study of abnormal scale in such uncommon patients may provide crucial clues to the biochemistry of normal epidermal differentiation, and to the construction of a normal, functional stratum corneum.

## What Promotes Adhesions of Leukocytes to Endothelium in Skin Diseases?

Why do white blood cells stick to the vascular endothelium, enter tissue, and cause inflammatory damage? It is now clear that one major factor is the presence of adhesion molecules which promote the attachment of leukocytes to other cells and to the extracellular matrix. At present count there are four major groups of adhesion molecules which promote leukocyte attachment to endothelium: LEC-CAM (e.g., ELAM-1), Hermes 1, ICAM-1, and INCAM. In this issue are two papers which deal with leukocyte attachment to endothelial cells using two very different experimental approaches.

George Murphy, Wendy Matis, and Robert Lavker at the University of Pennsylvania have shown that the neuropeptide substance P can induce expression of the endothelial adhesion molecule ELAM-1 on endothelial cells in explant cultures of human skin. This event is associated with mast cell degranulation and can be blocked by pretreatment of the explant tissue with inhibitors of mast cell degranulation such as cromolyn. According to Murphy, "This research was begun to determine whether there was functional significance to the small myelinated nerve fibers associated with mast cell membranes." He feels that substance P released from such nerve endings stimulates mast cell degranulation, and that mast cell products induce ELAM-1. This molecule might then promote leukocyte attachment to the endothelium and initiate leukocyte

0022-202X/90/$3.50 